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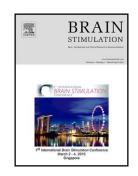
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Title: The effects of direct current stimulation on exercise performance, pacing and perception in temperate and hot environments

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#### **Highlights**

- Anodal tDCS has shown promise as an intervention to lower perceived exertion during exercise at low exercise intensities.
- The effects of tDCS on an ecologically valid self-paced exercise task have yet to be examined at high intensities in temperate conditions when contrasted to a valid SHAM control.
- Similarly, the effects of tDCS have not been examined on perception and performance in hot environments.
- Two studies were conducted but showed perception and performance were unaltered by tDCS.
- Anodal tDCS does not influence high intensity exercise performance and perception in temperate or hot conditions.
- Anodal tDCS may only enhance exercise perception and performance at low exercise intensities before full parasympathetic withdrawal has taken place.

#### **ABSTRACT**

**Background.** Transcranial direct current stimulation (tDCS) is a non-invasive neuromodulatory technique and has previously been shown to enhance submaximal exercise by reducing rating of perceived exertion (RPE). The present study examined the effects of tDCS on high-intensity self-paced exercise in temperate conditions and fixed followed by maximal exercise in the heat; it was hypothesised performance and RPE would be altered.

**Methods.** Two separate studies were undertaken in which exercise was preceded by 20-minutes of sham tDCS (SHAM), or anodal tDCS (TDCS). Study 1: six males completed a 20-km cycling time trial, on two occasions. Power output (PO), RPE, O<sub>2</sub> pulse, and heart rate (HR) were measured throughout. Study 2: eight males completed fixed intensity cycling exercise at 55% of a pre-determined maximal power output (P<sub>Max</sub>) for 25-minutes before undertaking a time to exhaustion test (TTE; 75% P<sub>Max</sub>) in hot conditions (33°C), on two occasions. Test duration, heart rate, thermal and perceptual responses were measured. Study specific and combined statistical analyses was undertaken and effect sizes established..

**Results.** Study 1: mean PO was not improved with the tDCS (197  $\pm$  20 W) compared to SHAM (197  $\pm$  12 W) and there were no differences in pacing profile HR, O<sub>2</sub> pulse or RPE (p > .05). Study 2: TTE duration (SHAM 314  $\pm$  334 s cf 237  $\pm$  362 s tDCS), thermal, heart rate and perceptual responses were unchanged by tDCS compared to SHAM (p > .05). When combined, performance in the SHAM trial tended to better than the tDCS.

**Conclusion.** tDCS did not influence cycling performance (study 1) exercise tolerance (study 2) or perception (studies 1&2). tDCS does not appear to facilitate high intensity exercise performance or exercise performance in the heat.

**Keywords**. Anodal stimulation, fixed and self-paced exercise, environmental temperature.

#### **INTRODUCTION**

Transcranial direct current stimulation (tDCS) is a non-invasive technique for modulating brain function[1] and works by passing a mild electrical current over the scalp, altering neuronal excitability[2]. The change in excitability is dependent on both the placement and polarity of the stimulation[3]. Anodal stimulation results in a depolarisation of resting membrane potential, increasing spontaneous neural firing, whereas cathodal stimulation results in a hyperpolarisation of resting membrane potential, depressing neuronal excitability. Acute exposure to tDCS of as little as 10-minutes has been shown to result in changes in regional neuronal excitability lasting for 50-minutes or greater[4]. Clinical studies suggest that transiently altering aspects of brain function using tDCS may provide a number of potential benefits, dependent upon the area of stimulation, including the facilitation of learning[5,6], inducing analgesia[7,8], and in treatment of Parkinson's disease[9] and Alzheimer's disease[10].

There is also some evidence that tDCS may provide a beneficial effect on exercise performance[11,12,13]. In particular, Okano *et al*[12] reported significant reductions in both heart rate and rating of perceived exertion (RPE) at sub-maximal, but not maximal intensities in trained cyclists. Okano *et al*[12] also noted there were moderate improvements in peak power output (~4%) during a maximal incremental exercise test. This occurred following 20-minutes of anodal tDCS at 2 mA over the left temporal cortex (TC), which likely increased neuronal excitability of the insular cortex (IC) as well as influencing collateral regions such as the left TC and frontal lobe. Stimulation of the IC may be important as this region is involved in the awareness of subjective feelings of the body, related to athlete's perceptions of physical

exertion during dynamic exercise[14,15]. The associated performance effects have also been shown by Cogiamanian *et al*[11] and Tanaka *et al*[13] who demonstrated significant improvements in time-to-exhaustion after tDCS of ~35% in an isometric arm endurance task, whereas Tanaka *et al*.[13] reported significant improvements in maximal pinch force of the left hallux compared with sham and cathodal stimulation conditions. More recently, Angius *et al*.[16] have studied maximal exercise and pain tolerance and reported improved pain tolerance during a cold pressor test but saw no beneficial effect on time to exhaustion (TTE) albeit after only 10-minutes of tDCS stimulation. The findings of these studies have interesting implications concerning the potential benefits of tDCS on 'real world' exercise performance for example during self-paced exercise using multiple muscle groups, or exercise performance in the heat.

During many exercise tasks the primary aim is to complete a given distance in the shortest time possible. To achieve this, athletes regulate their energetic resources to maintain a maximal sustainable intensity to avoid premature fatigue and exhaustion[17]; this is termed pacing. The role of the brain in pacing is not entirely clear although RPE, which has been shown to be modulated by tDCS, is involved as a key perceptual anchor for the regulation and distribution of effort[18,19]. Manipulating perceived exertion might therefore provide a potential mechanism for influencing exercise pacing and performance and this may also extend to other environments.

Exercise in hot conditions is also profoundly influenced by perceived exertion. Hot conditions are known to accelerate the rate of rise in RPE during fixed intensity (FI) exercise when contrasted to cool conditions[20]. Theoretically raised deep body temperature (hyperthermia) causes an increased demand of the central nervous system to recruit motor units to generate muscular force[19]. When power output is fixed at a high intensity, such as during a TTE, an increased level of effort is required compared to that of cool conditions [20]. Therefore, unless power output is allowed to drop, exercise is terminated prematurely due to

unsustainably high levels of exertion. Even at sub-maximal intensities RPE is likely to be raised by hot conditions and may influence subsequent maximal exercise.

There is a plausible rationale for anodal tDCS to enhance both self-paced exercise performance and exercise performance in the heat; accordingly, two studies were undertaken. The purpose of study one was to examine the effect of anodal tDCS on performance, pacing, and the associated physiological and perceptual responses during a self-paced exercise task. It was hypothesised that anodal tDCS would alter exercise pacing and be ergogenic. It was postulated that tDCS would alter relationship between RPE and work rate, resulting in a higher mean power output. The purpose of study two was to examine the performance and perceptual consequences of tDCS during rest, sub-maximal fixed intensity exercise and during a subsequent TTE in hot conditions. Fixing work rate would provide a defined intensity against which changes in RPE induced by tDCS could be discerned. It was hypothesised that RPE would be lower after tDCS during sub-maximal fixed intensity exercise and that TTE would be extended when contrasted to sham control. The differing methods of these two studies were employed to isolate the mechanism by which tDCS may work upon RPE and power output.

#### MATERIALS AND METHODS

#### Study 1 & 2 common features

#### **Participants**

The studies were approved by their institutional ethics committees and participants gave their written informed consent. Participants undertook regular habitual exercise (≥ 150-minutes of exercise per week) and were accustomed to exercise of a maximal nature, although were not trained cyclists. Tests were conducted at the same time of day with standardised pre-test procedures.

#### **Experimental Design**

Stimulation conditions (either tDCS or SHAM) were counterbalanced and blinded with the stimulation condition determined by a third party individual. To reduce participant vigilance participants were informed that they would be receiving the tDCS treatment on both occasions. All visits were separated by a minimum of 48 h.

#### Stimulation procedures

The tDCS procedures were administered by trained personnel. Similar to the procedures of Okano[12], the anode electrode was placed over the T3 area of the skull corresponding to the TC, located 40% of the distance on the left from the vertex (Cz) to the tragus, according to the international standards for the EEG 10-20 system[21]. The cathode electrode was placed over the contralateral supraorbital area (Fp2). Stimulation was provided for a period of 20-minutes (tDCS trial only), applied through a pair of sponges (3.5 cm<sup>2</sup> in study 1 and 4.5 cm<sup>2</sup> in study 2) pre-soaked in saline solution. Participants received a constant electric current at 1.5 mA in study 1 and 2.0 mA in study 2 culminating in similar current densities; i.e. 0.43 mA·cm<sup>-2</sup> & 0.44 mA·cm<sup>-2</sup>. Stimulation was delivered using battery powered DC stimulators (Study 1: CX6650, Rolf Schneider Electronics, Gleichen, Germany; Study 2: HDCstim DC stimulator, Newronika S.r.l, Milan, Italy). For the SHAM condition electrodes were placed in the same position as for the tDCS condition. However, the stimulator was turned off after 30-seconds of stimulation. This method replicates the sensory feelings experienced in the tDCS trial (i.e. itching and tingling sensations) and cannot be distinguished from it whether the stimulation is continued or stopped[22]. Following study completion participants were required to report whether they were able to detect any deception. At the end of each exercise test, a fingertip blood sample was taken to establish post-exercise blood lactate concentration (Biosen, C-line Sport, Germany).

#### **Procedures**

All testing was undertaken on a Velotron Dynafit Pro cycling ergometer (RacerMate Inc., Seattle, WA, USA). Velotron 3D software (RacerMate Inc., Seattle, USA) was used to construct the flat 20 km virtual TT course used in study 1. In study 2 the manual ergometer function was used to administer the power output increments during all tests. In both studies the individual positional adjustments (saddle and handlebar position) were prescribed by the participant in the first trial and replicated thereafter.

Study 1 – Effect of TDCS on pacing, perception and performance during self-paced exercise in temperate conditions

#### **Participants**

Six male participants (age  $21 \pm 2$  years, height  $1.85 \pm 0.06$  m, mass  $80.3 \pm 10.4$  kg, maximal aerobic capacity  $3.87 \pm 0.56$  L·min<sup>-1</sup>) were recruited.

#### **Experimental design**

Participants visited the laboratory on four separate occasions. During the first visit, participants undertook a graded exercise test to exhaustion (GXT) to determine maximal aerobic capacity. In the following three visits participants undertook a 20 km cycling time trial (TT). Visit 2 (FAM) was a familiarisation TT. Visits 3 and 4 were experimental trials with participants exposed to either 20-minutes of anodal tDCS (tDCS) or 20-minutes of sham anodal tDCS (SHAM) prior to the TT start. Testing was conducted in an air-conditioned laboratory  $(21 \pm 1^{\circ}\text{C}, 38 \pm 4\%\text{RH})$ .

#### **Procedures**

#### **Graded Exercise Test**

The GXT commenced at 60 W and used step increases of 25 W·min<sup>-1</sup>. The test was terminated when participants were unable maintain a cadence within 10 revs·min<sup>-1</sup> of the

target cadence (70 revs·min<sup>-1</sup>), or on volitional exhaustion. During the GXT, participants wore a heart rate monitor recording heart rate at 15 s intervals (Polar RS800, Electro OY, Polar, Warwick, UK) and were connected by facemask to an online gas analysis system (Quark B2, Cosmed, Italy) to establish maximal aerobic capacity ( $\dot{V}O_{2max}$ ).

#### 20 km TT: familiarisation and experimental trials

Thirty minutes prior to commencing each TT, participants completed a 10-minute warm-up at 100 W. Thereafter, participants were exposed to 20-minutes of either anodal tDCS or SHAM stimulation whilst seated on the bike. Subsequently the stimulation pads were removed. Before commencing exercise, participants were instructed to complete the TT in the fastest time possible. During the TT participants received feedback of distance covered but all other feedback was occluded and no verbal encouragement was provided. For the duration of the TT, participants were required to wear a heart rate monitor and were connected to the gas analysis system,. Rating of perceived exertion (RPE;[23]) was obtained every 4 km of the TT. Participants exercised in front of a fan providing convective airflow at a speed of 2.13 m·s<sup>-1</sup>. A blood sample was drawn by finger prick lancet on completion of each 4 km segment to establish the blood lactate concentration profile throughout the TT

Study 2 – Effect of tDCS on perception and thermal responses during fixed intensity exercise in hot conditions

#### **Participants**

Eight male participants (age 21  $\pm$  1 years, height 1.76  $\pm$  0.05 m, mass 86.4  $\pm$  15.0 kg, maximal power output ( $P_{Max}$  235  $\pm$  13 W) were recruited.

#### **Experimental design**

Participants visited the laboratory on three occasions. During the first visit, participants undertook a graded exercise test to exhaustion to establish maximal power output ( $P_{Max}$ ). In the following two visits participants they were exposed to hot (33°C; 20% relative humidity [RH]) conditions whilst in an environmental chamber. They first undertook a 20-minute rest period during which SHAM or tDCS were administered followed by a 25-minute period of fixed intensity (FI) cycling exercise at 55% of  $P_{Max}$  and concluding with a test to exhaustion (TTE) at 75% of  $P_{Max}$ .

#### **Procedures**

#### Power Max Test

The  $P_{Max}$  was completed in cool conditions 16 °C 40% RH. Having warmed up the  $P_{Max}$  commenced at 50 W and used step increases of 25 W·min<sup>-1</sup>.  $P_{Max}$  was terminated when participants were unable maintain a cadence within 10 revs·min<sup>-1</sup> of the target cadence (70 revs·min<sup>-1</sup>), or on volitional exhaustion.  $P_{Max}$  was established objectively as the highest power output that was sustained for a minimum 15-second period.

#### Rest, FI and TTE in hot conditions

Following their arrival at the environmental chamber participants changed in to their cycling clothing (anklet socks, shorts, and running trainers; the same clothing was worn on each occasion). They were then instrumented with a heart rate monitor (Polar FT1, Polar Electro Oy, Kempele, Finland) and a calibrated aural thermistor ( $T_{au}$ ) was self-inserted in to the external auditory meatus, insulated with cotton wool and neoprene. Thereafter, six skin thermistors, were attached on the left hand side of the body at the bicep, chest, subscapular, forearm, thigh and hamstring to enable the calculation of mean skin temperature ( $T_{msk}$ [24]). Participants were also introduced to the perceptual scales to measure thermal sensation (TS), thermal comfort (TC) and RPE. TS and TC were measured using visual analogue scales; TS

ranged from 0 cm – *very cold* to 20 cm – *very hot*, TS ranged from 0 cm – *very uncomfortable* to 20 cm – *very comfortable* where the worded descriptors act as a guide only. RPE was measured using a 15-point category ratio scale[25].

Once instrumented, participants entered the environmental chamber and were seated on a chair to undertake a 20-minute rest period during which they received tDCS or the SHAM treatment. The tDCS pads were then removed and the participant mounted the cycle ergometer; a 3-minute transition period. They then commenced FI exercise for 25-minutes following which they transitioned to the TTE at 75% of  $P_{Max}$  using 10 W increments every 5 seconds until the target wattage was reached. Participants reported their perceptual experiences of TS, TC and RPE every 5-minutes at rest and throughout the FI and TTE. Once volitional exhaustion was reached, (same termination criteria as  $P_{Max}$  tests), data logging systems were stopped, they pedalled briefly against a low fixed resistance and then exited the environmental chamber. During the TTE, participants did not receive any temporal feedback or verbal encouragement. Throughout all trials participants exercised in front of a fan providing convective airflow at a speed of  $2.13 \text{ m} \cdot \text{s}^{-1}$ .

#### **Data Analysis**

Normality was assessed using Kolmogorov-Smirnov test. Comparisons were made within participant, between condition and across time using repeated measures analysis of variance (ANOVA; condition [2] x time [5,6, or 8 points]; see study specific details). Sphericity was examined using Mauchley's test and were adjusted using Greenhouse-Geisser where necessary. *Post-hoc* statistically significant effects were determined using pair-wise comparisons with Bonferroni correction. Alpha level was set at 0.05 for all tests. Effect sizes (ES) were calculated using Cohen's *d* and interpreted according to Hopkins[26]. Data are

presented as mean  $\pm$  SD. All statistical tests were conducted using SPSS version 21.0 (IBM, Armonk, NY, USA).

#### Study 1

Mean trial power output (PO) was used to examine cycling performance. PO, heart rate (HR), and RPE for each TT were allocated into sequential 4 km 'bins'. Cardiac efficiency was determined using the  $O_2$  pulse method (i.e.  $\dot{V}O_{2max}$  /heart rate at  $\dot{V}O_{2max}$ ; see Heath *et al.*, 1981), averaged over each 4 km bin; providing 5 time points for ANOVA.

#### Study 2

Mean  $\pm$  SD were calculated for  $\Delta T_{au}$ ,  $T_{msk}$ , HR, TC, and TS after 10 and 20 minutes of rest, for every 5-minutes of the FI period and at the end of the TTE (8 time points). RPE was examined at 5-minute intervals during the FI period and at the start and end of the TTE (6 time points).

#### Studies 1 & 2 - Combined Analysis

Trial completion times (including the FI period in study 2), terminal RPE and post-exercise blood lactate concentration from the TT in study 1 and the TTE in study 2 were pooled into the tDCS and SHAM conditions from the respective studies. They were compared between-conditions using a Wilcoxon signed ranks test. This enabled the polar nature of the performance test in study 1 (i.e. a shorter trial indicated a better performance) and study 2 (i.e. a longer test indicated a better performance) to be accounted for by attributing a positive rank to the test performance from study 1 when TT performances were quicker in the SHAM treatment to calculate the performance statistic.

#### RESULTS

None of the participants in study 1 or 2 reported any awareness of the treatment deception.

#### Study 1

#### **Performance**

Mean PO was  $197 \pm 12$  W and  $197 \pm 20$  W for SHAM and tDCS respectively which were not significantly different between conditions ( $F_{(2, 10)} = .920$ ; p = .430). Mean TT completion time was  $2181 \pm 56$  s (i.e. 36 min 21 s  $\pm 56$  s) and  $2181 \pm 88$  s (i.e. 36 min 21 s  $\pm 88$  s) in the SHAM and tDCS respectively.

#### **Pacing**

The PO profile showed a consistent pattern across 4 km 'bins' (see Figure 1A) and this did not culminate in a significant difference between conditions ( $F_{(1, 5)} = .001$ ; p = .993), or over the TT distance ( $F_{(4,20)} = 1.709$ ; p = .187), indicating an even distribution of PO across the TT (no interaction effect:  $F_{(4, 20)} = .438$ ; p = .780) effect size estimates revealed a trivial effect (d = .002).

#### \*\*\*INSERT FIGURE 1 NEAR HERE\*\*\*

#### Cardiac responses

#### Heart rate

No significant differences were observed between conditions ( $F_{(1,5)} = .109$ ; p = .755) and ES were small to trivial (d = .108). Further analysis did a reveal a significant effect of trial distance on HR increasing as the trial ensued ( $F_{(4, 20)} = 14.078$ ; p < .001); see Figure 1B. Terminal HR also indicated an increase in intensity towards the end of the TT, with peak HR achieved at the 20 km distance point; there was no significant interaction effect ( $F_{(4,20)} = .479$ ; p = .751).

#### Cardiac efficiency

The serial pattern of  $O_2$  pulse between conditions is shown in Table 1. No significant differences were observed between conditions ( $F_{(1,5)} = .228$ ; p = .653). ES were small to trivial between the tDCS and SHAM (d = .102). No significant differences were observed with regard to either distance ( $F_{(4,20)} = .880$ ; p = .493) or interaction effect between condition and distance ( $F_{(4,20)} = 1.805$ ; p = .167).

\*\*\*INSERT TABLE 1 NEAR HERE\*\*\*

#### **Blood lactate**

The serial pattern of [BLa] between conditions is shown in Table 1. Both conditions induced changes in [BLa] but not to any differing extent in either condition ( $F_{(1,5)} = 3.767$ ; p = .110). ES revealed trivial to small effects (d = .299). Examination of the effect of distance on [BLa] revealed a significant difference increasing as the trial ensued ( $F_{(1.336, 6.680)} = 11.363$ ; p = .010); see Table 1. There was also no effect ( $F_{(4,20)} = 1.195$ ; p = .344). Mean post trial [BLa] was  $9.96 \pm 3.29$  mmol.L<sup>-1</sup> and  $8.08 \pm 3.21$  mmol.L<sup>-1</sup> in the SHAM and tDCS respectively.

#### **Perceptual responses**

The serial pattern of RPE between conditions is shown below (Figure 1C). RPE was similar in each condition ( $F_{(1,5)} = 3.270$ ; p = .130). Examination of ES between conditions revealed small to trivial effects between the tDCS and SHAM (d = .225). Significant differences in RPE were evident as the TT ensued with RPE increasing to a peak of  $19 \pm 2$  and  $18 \pm 2$  in the

SHAM and tDCS respectively ( $F_{(4, 20)} = 47.247$ ; p = .001); see Figure 1C. No significant interaction effects on RPE were observed ( $F_{(4,20)} = 1.194$ ; p = .344).

#### Study 2

#### **Performance Data**

Mean TTE was  $314 \pm 334$  s and  $237 \pm 362$  s in the SHAM and tDCS, respectively.

#### **Aural Temperature**

The aural temperature response for one participant was incomplete for one test; therefore n=7 for the deep body temperature responses. The aural temperature responses were similar between condition throughout the rest, FI and TTE periods of the SHAM and tDCS trials and had changed by a similar magnitude by the end of each trial (SHAM +1.19  $\pm$  0.9°C; tDCS +1.03  $\pm$  0.2°C; no main effect for condition: ( $F_{(1,6)}=1.472$ ; p=.271). The aural temperature rose steadily throughout the rest and exercise periods culminating in an effect over time ( $F_{(7,42)}=27.077$ ; p=.001) but this did not happen to any differing extent in either the SHAM or the tDCS condition (no main effect or interaction effect:  $F_{(7,42)}=.431$ ; p=.877). A small effect size was observed (d=.27);  $T_{au}$  data are displayed in Figure 2A.

# \*\*\*INSERT FIGURE 2 NEAR HERE\*\*\*

#### **Mean Skin Temperature**

The  $T_{msk}$  responses were also similar and were closely matched in the SHAM compared to the tDCS condition (no main effect for condition: ( $F_{(1,7)} = .165$ ; p = .696). The  $T_{msk}$  increased steadily throughout the trial (main effect for time:  $F_{(7,49)} = .77.357$ ; p = .001) but reached similar absolute levels in both the SHAM and tDCS conditions (SHAM 35.17  $\pm$  0.4°C; tDCS

 $35.09 \pm 0.3$ °C; no interaction effect:  $F_{(7,49)} = .639$ ; p = .721). A small to trivial effect size was observed (d = .11);  $T_{msk}$  data are displayed in Figure 2B.

#### **Heart Rate Response**

The HR responses were similar between conditions throughout rest, FI exercise and the TTE (no main effect for condition:  $(F_{(1,7)} = 1.886; p = .212)$  although HR data were not complete at one time point (i.e., start FI period) so only 7 points were analysed. The HR response increased steadily throughout the FI exercise period indicating some cardiovascular drift (main effect for time:  $F_{(7,49)} = 231.567; p = .001$ ) but this did not differ between condition with similar terminal heart rates (SHAM 178  $\pm$  11 b·min<sup>-1</sup>; tDCS 170  $\pm$  11 b·min<sup>-1</sup>; no interaction effect:  $F_{(7,49)} = .854; p = .536$ ). A trivial effect size was observed (d = .07); HR data are displayed in Figure 2C.

#### RPE, TS and TC Responses

RPE data during the FI period were similar in each condition and were associated with the worded descriptor *hard* to *very hard* by the end of the FI period. Yet, this did not differ between condition (no main effect for condition:  $F_{(1,7)} = 3.84$ ; p = .555). There were similar terminal RPE ratings at the end of the FI in both conditions SHAM (16 ± 3) and tDCS conditions (17 ± 2; no interaction effect:  $F_{(7,49)} = 1.236$ ; p = .313). There was evidence of continued increase in perceived exertion as the trial ensued despite the fixed work rate which is a further indicator of increasing thermal strain and cardiovascular drift (time effect:  $F_{(7,49)} = 63.804$ ; p = .001). A trivial effect size was observed (d = .14).

After FI exercise participant's mean TS was *hot* but not to any differing extent in either condition (no main effect for condition:  $F_{(1,7)} = .070$ ; p = .800). The mean TC response was *uncomfortable* before the TTE was undertaken and was similar in each condition (no main

effect for condition:  $F_{(1,7)} = .002$ ; p = .963). As the trial ensued both the TS and TC responses increased (TS time effect:  $F_{(7,49)} = 30.618$ ; p = .001; TC time effect:  $F_{(7,49)} = 29.857$ ; p = .001). After the TTE was complete there was no difference in the associated worded descriptors (no interaction effect: TS  $F_{(7,49)} = 1.934$ ; p = .084; no interaction effect:  $F_{(7,49)} = .659$ ; p = .705). Trivial to small effect sizes were observed for TS (d = .16) and TC (d = .18); see Figure 3.

#### \*\*\*INSERT FIGURE 3 NEAR HERE\*\*\*

#### **Combined Analysis**

Wilcoxon signed ranks test indicated no difference in performance between the SHAM and tDCS trials (Z = -1.161, p = 0.245). Terminal RPE was  $19 \pm 2$  and  $18 \pm 2$  at the end of the trial in the SHAM and tDCS conditions but were not different (Z = -1.350, p = .177) and produced a small effect size (d = .27). [BLa] was significantly higher at the end of the trial in SHAM treatment condition ( $6.20 \pm 4.0 \text{ mmol}\cdot\text{L}^{-1}$ ) compared to the tDCS condition ( $5.08 \pm 3.5 \text{ mmol}\cdot\text{L}^{-1}$ ) by an average of  $1.07 \pm 1.0 \text{ mmol}\cdot\text{L}^{-1}$  (Z = 2.543, z = .011) although this effect size was small (z = .30).

#### **DISCUSSION**

The aim of study one was to examine the effect of anodal tDCS over the left TC on self-paced 20 km cycling time trial performance, relative to an appropriate sham treatment. The findings indicate that 20-minutes of anodal stimulation prior to exercise did not significantly improve cycling performance. Indeed the data show that the pacing profile, physiological and perceptual responses were conspicuously similar following anodal tDCS compared to the SHAM treatment.

The aim of study two was to examine the performance and perceptual consequences of tDCS during rest, sub-maximal fixed intensity exercise and during a subsequent time trial to exhaustion in hot conditions. We found no evidence of any alteration in thermal perception at rest, perceived exertion and physiological responses during sub-maximal exercise or any ergogenic effect during a subsequent TTE trial. Indeed, it seems that the perceptual and physiological responses during this part of the study were tightly regulated and not disturbed by anodal tDCS at any stage.

The absence of an improvement in performance resulting from tDCS contrasts with some previously reported results[11,12]. By contrast, our data agree with the recent work of Angius *et al.*[16] who examined a cycling TTE and found no beneficial effect. Collectively it appears that anodal tDCS prior to exercise does not improve dynamic exercise of a maximal nature whether it is at a fixed intensity or if it is self-paced. Indeed, enhanced performance appeared more likely in the SHAM treatment condition where post trial blood lactate was also higher than that seen in the tDCS trial. The evidence for a performance effect across published studies is not compelling unless an isometric muscle endurance exercise task is undertaken where an effect remains plausible[11], although the relevance of this model in a sporting context is limited.

An effect on perception was more likely[12,16]. As previous findings indicated that tDCS may provoke a dissociative effect between RPE and exercise work rate, which in self-

paced exercise may encourage the adoption of a higher PO for a given RPE [12,28]. Theoretically, during FI exercise, RPE would have been lower for a given sub-maximal PO yet there was no evidence of this in study 2. In particular, stimulation targeted to alter neuronal excitability of the IC appeared to provide a logical mechanism for manipulating RPE. However, the pacing (study 1 only) and RPE profiles (both studies) were almost identical between tDCS and SHAM conditions, both of which were associated with a consistent template (see Figure 1A & 1C). Similarities in exercise pacing and RPE appear to suggest that, in the present study, tDCS was insufficient in modulating the central mechanisms involved in the awareness of physiological strain.

Our study also extends the range of perceptual variables examined following tDCS stimulation to include thermal perception; a further novel aspect to our investigation. Given that Angius[16] reported a change in cold pain perception during a cold pressor task it is surprising that perception of thermal sensation and thermal comfort were not affected. In explaining this discrepancy it is possible that the beneficial effects of tDCS are specific to pain afferents rather than exclusively thermal afferents which are related but comprise different thermoreceptor network loops[29]. It is also possible that the benefits of tDCS are specific to cold thermal stimuli rather than hot as studied here. Lastly, the timing of tDCS stimulation may also explain this discrepancy at rest (study 2 only) as tDCS stimulation was being delivered during this period and it may take time for the neural network to become sufficiently stimulated to induce a change in perception. On balance there is no consistent evidence across the literature that there are cognitive changes induced by tDCS and these would form, in part, the internal verbal associations with altered thermal perceptions [30].

In addition to changes in RPE, previous studies have also reported reductions in HR, both at rest and at submaximal exercise intensities following stimulation[12,31]. Interestingly, with respect to Okano *et al.*[12], reductions in HR during exercise appeared to indicate possible improvements in cardiac efficiency. Changes in HR were attributed to increased

parasympathetic modulation. However, the results of the present study indicated that HR was not significantly affected by anodal tDCS. Similarly, cardiac efficiency, as indicated by O<sub>2</sub> pulse, was not significantly different between conditions. These findings may be explained by the effect of exercise intensity on the regulation of HR. Autonomic cardiac responses are regulated by both sympathetic and parasympathetic neural pathways[32]. Parasympathetic pathways regulate HR predominantly at rest and during low intensity exercise. However, at higher exercise intensities HR becomes principally regulated through sympathetic pathways [32]. The relatively short duration of the exercise task in study one likely encouraged the adoption of heavier exercise work rates, triggering a predominantly sympathetic HR response which may be unaffected by tDCS. It has previously been reported that at exercise intensities approaching VO<sub>2max</sub> tDCS does not provide a significant effect on HR compared with placebo[12]. In more prolonged exercise tasks, where self-selected work rates are lower, tDCS may provide a more noticeable effect on HR. However, this was not evident at the submaximal intensity used in study 2 either, although this may also have taken place at an intensity above the point of full parasympathetic withdrawal. The effect of tDCS over prolonged duration exercise beyond that studied here may be minimal considering the influence of tDCS is transient and typically subsides after ~ 30 to 60 minutes[13,6].

The disparity in the findings between the present study and previous exercise-based tDCS studies may also be explained by differences in the 'blinding' procedures used to disguise stimulation condition. Indeed, it was recently concluded that effective participant and experimenter blinding was difficult to achieve with some of the tDCS procedures documented in the open literature [34]. This may apply to studies that have examined exercise or muscular performance tests following tDCS treatment. For example, Cogiamanian *et al*[11] compared performances between stimulation and control groups, without a consideration for a possible placebo effect. Whereas, Okano *et al.*[12] did use a sham stimulation condition for comparison; however, the authors reported that subjects were fully informed of the study's

methodology. Angius *et al*[16] used a suitable control in addition to the SHAM and tDCS procedures and only saw a beneficial effect on pain perception. We propose that the double blinding procedures used in the present studies provide a more effective deception, since participants were encouraged to believe that they received identical treatment on both occasions, thereby reducing vigilance for the treatment condition. Importantly, our post-study follow up procedures did not reveal the participant's ability to discern between the SHAM and tDCS protocols which has been documented to occur above the probability of chance detection in other studies where blinding was clearly not achieved [34]. Indeed, the tendency for participants to be better in the SHAM trial may indicate some contralateral interference from the cathode electrode on the adjacent cerebral hemispheres and this would have been lesser in the SHAM trial [35]. However, others[12] have seen differences with the methodological approach we employed here.

The present studies were not without limitation. Indeed both studies used a low number of participants. Small sample sizes can limit the statistical power of the data that is collected, increasing the possibility of a type II statistical error. However, our expectation that performance would be improved with tDCS (i.e. a one tail hypothesis) would not be supported assuming the tendency to be better in the SHAM trial prevailed with a larger number of participants. It is important to note that our participant numbers exceeded other exercise studies that have independently observed[12] and failed to observe[16] performance effects using similar exercise protocols. This suggests that stimulation is unlikely to provide any clinically significant benefit to 'real world' exercise performance. We therefore contend that anodal tDCS may not induce sufficiently large changes in neuronal activity to influence high-intensity exercise performance. Indeed, the size of the effect of tDCS on neuronal excitability is reportedly relatively small compared with other neuro-modulatory techniques such as transcranial magnetic stimulation[TMS, 33]. Meta-analyses data support the idea that there is large inter-participant variability in the neuronal response to tDCS stimulation and the

neurophysiological effect of tDCS may be restricted to modulation of motor evoked potential (MEP) as estimated by TMS [36]. In the present study, since both HR and RPE are associated with the IC, a lack of change in both variables implies that neuronal excitability in the IC was not sufficiently altered following stimulation to result in a meaningful effect. Investigation using neuroimaging techniques such as electroencephalography or functional magnetic resonance imaging might substantiate these postulations [35]. Similarly, the inclusion of a control site for tDCS stimulation and a control task may have enhanced the mechanistic precision with which we could discuss our findings [35].

In summary, the present investigations are the first to examine the effects of anodal tDCS on self-paced exercise performance and exercise performance in the heat. The results indicate that the stimulus provided by tDCS was insufficient to induce significant improvements in high-intensity exercise performance in temperate or hot conditions.

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#### **Figure Legends**

**Figure 1.** Mean  $\pm$  SD serial pattern of power output (1A.), heart rate (1B.) and RPE (1C.) throughout the TT following the SHAM and tDCS treatment interventions; \* indicates difference between sequential data points, n = 6.

**Figure 2.** Mean  $\pm$  SD aural temperature change (2A.), mean skin temperature response (2B.) and heart rate response (2C.) at rest, during the FI period and at the start and end of the TTE in the SHAM and tDCS treatment interventions; \* indicates difference between sequential data points, n = 8.

**Figure 3.** Mean  $\pm$  SD RPE (3A.), TS (3B.) and TC (3C.) at rest, during the FI period and at the start and end of the TTE in the SHAM and tDCS treatment interventions; \* indicates difference between sequential data points, n = 8.

#### **Tables**

**Table 1. Mean \pm SD** BLa and O<sub>2</sub> Pulse for every 4 km completed of the TT in the SHAM and TDCS conditions in study 1; \* indicates difference between sequential data points, n = 6.

	4 km	8 km	12 km	16 km	20 km
SHAM	70				
	6.27	6.49	6.66	7.16	9.96
BLa					
( N# 17-1)	±2.58	$\pm 2.68$	$\pm 2.46$	$\pm 2.98$	±3.29
(mMol.L <sup>-1</sup> )					
TDCS					
	5.28	6.00	6.01	6.25	8.08*
BLa					
( 1\	$\pm 2.72$	±3.15	±3.04	$\pm 2.69$	±3.21
(mMol.L <sup>-1</sup> )					
SHAM O <sub>2</sub>	2				
•	17.67	17.26	17.35	18.36	19.08
Pulse					
1	$\pm 3.08$	$\pm 2.67$	$\pm 2.49$	$\pm 3.42$	$\pm 3.80$
$(\mathbf{mL.b}^{-1})$					

TDCS	$O_2$ 19.07	18.28	17.79	17.93	18.37	
Pulse	±3.64	±2.10	±2.14	±1.93	±2.69	
$(\mathbf{mL.b}^{-1})$	_5.01	_2.10	_2	_1.,,		